REMARKS/ARGUMENTS

The undersigned gratefully acknowledges and thanks the Examiner for the time and courtesy extended during the telephonic interview which occurred on December 14, 2007. In light of the above amendments and the following remarks, prepared in accordance with the discussions, it is respectfully submitted that all pending claims are now in condition for allowance. The Examiner is respectfully invited to contact the undersigned if any issues remain pending.

Pending claims 1-7, 12-18, 21, 23, 25 and 34-38 stand rejected under 35 U.S.C. §103(a) over U.S. Patent No. 6,321,105 (Jenkins) in view of U.S. Patent No. 6,104,943 (Frederick). Applicants respectfully traverse the rejection. As to claim 1, Jenkins nowhere teaches or suggests directly detecting regional neural activity from and concurrently with transient magnetic fields induced by the regional neural activity, where the direct detection is without measurement of hemodynamic or metabolic changes and which occurs in a time window before hemodynamic activity as a result of the regional neural activity. Instead, Jenkins merely teaches the conventional manner of performing MRI data acquisition and analysis based on a hemodynamic response invoked by neuronal activation. Jenkins, col. 1, lns. 51-54. Thus Jenkins maps neurotransmitter activity based on metabolic response (*id.*, col. 2, lns. 60-66), not from and concurrently with transient magnetic fields induced by regional neural activity. Furthermore, the mapping performed in Jenkins is done based on metabolic response that follows a diagnostic challenge. In other words, relative cerebral blood volume (rCBV) "changes *following* diagnostic challenge..." Jenkins, 3:8-9 (emphasis added). That is, Jenkins teaches "the metabolic response associated with neuronal activation following diagnostic challenge results in a substantial increase in rCBV and hence a decrease in Signal Intensity (SI)." Jenkins, 2:60-66. Here, claim 1 recites a direct detection; in other words, changes in neuronal activity are detected via the magnetic field effects of neuronal firing *per se*, and not through hemodynamic or metabolic changes of hemodynamics which these changes in neuronal activity trigger, as taught by Jenkins (3:7-10) and contrasted by claim 1. As such, Jenkins does not map directly from and concurrently with transient magnetic fields induced by regional neural activity. Furthermore, Frederick fails to teach this missing subject matter from Jenkins. For at least these reasons, claim 1 and its dependent claims are patentable.

As to claim 14, nowhere does Jenkins teach or suggest directly mapping electromagnetic activity of a subject via magnetic resonance imaging. Certainly, Jenkins does not perform such direct mapping without a temporal delay from the electromagnetic activity, and where the direct mapping directly detects regional neural activity from and concurrently with the electromagnetic activity and without measurement of hemodynamic or metabolic changes. That is, as discussed above Jenkins teaches that its monitoring of rCBV changes follows diagnostic challenge and thus after a temporal delay. Jenkins thus indirectly maps neurotransmitter activity based on metabolic responses, not directly from electromagnetic activity. Accordingly, claim 14 and its dependent claims are patentable over the proposed combination, as Frederick fails to add anything in this regard. For at least similar reasons as to claims 1 and 14, independent claim 23 and the claims depending therefrom are also patentable. Note also as to claim 23, Jenkins infers neural activity from hemodynamic changes, and also fails to directly detect regional neural activity in a time window prior to hemodynamic activity, in contrast to the recited subject matter. Jenkins, 2:60 – 3:11. Since Frederick fails to teach anything in this regard, claim 23 and its dependent claims are patentable over the cited art.

Regarding independent claim 34, both Jenkins and Frederick fail to teach or suggest receiving MR signals from a subject prior to hemodynamic changes as a result of neuronal activity. In addition, the cited art fails to teach that the neuronal activity is measured from and concurrently with neural electromagnetic changes instead of from hemodynamic or metabolic changes induced by the neural electromagnetic changes. This is so, at least for the same reasons discussed above regarding claim 1. Accordingly, claim 34 and the claims depending therefrom are patentable over the cited art.

Pending claims 26-32 stand rejected under 35 U.S.C. §102(e) over U.S. Patent No. 6,477,399 (Biswal). Applicants respectfully traverse the rejection. As to claim 26, Biswal nowhere teaches a controller that directly detects a magnitude of magnetic resonance signals that represent a neuronal magnetic field, without measurement of hemodynamic or metabolic changes resulting from the regional neural activity and further where the direct detection occurs in a time window before hemodynamic activity. Instead, Biswal is directed to a conventional fMRI system (*e.g.*, Biswal, col. 6, lns. 57-62) in which brain activity is detected via hemodynamic and metabolic responses, rather than magnitude signals representing a neuronal magnetic field that is

induced by neural activity. *E.g.*, *id.* at col. 2, lns. 15-25. Accordingly, claims 26-32 are patentable.

For at least the same reasons as the independent claims from which they depend, the rejection of claims 9-11 and 19-20 under §103(a) over Jenkins and Frederick in view of Mueller is also overcome.

New claim 41 is patentable at least for the same reasons as claim 1 from which it depends.

In view of these remarks, the application is now in condition for allowance and the Examiner's prompt action in accordance therewith is respectfully requested. The Commissioner is authorized to charge any additional fees or credit any overpayment to Deposit Account No. 20-1504.

Respectfully submitted,

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